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Toxicological Profile for

LEAD

Draft for Public Comment

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U.S. DEPARTMENT OF HEALTH & HUMAN SERVICES
Public Health Service
Agency for Toxic Substances and Disease Registry

DRAFT

TOXICOLOGICAL PROFILE FOR LEAD

Prepared by:

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DISCLAIMER

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UPDATE STATEMENT

A Toxicological Profile for lead was released in April 1993. This edition supersedes any previously released draft or final profile.

Toxicological profiles are revised and republished as necessary, but no less than once every three years. For information regarding the update status of previously released profiles, contact ATSDR at:

Agency for Toxic Substances and Disease Registry
Division of Toxicology/Toxicology Information Branch
1600 Clifton Road NE, E-29
Atlanta, Georgia 30333

FOREWORD

This toxicological profile is prepared in accordance with guidelines developed by the Agency for Toxic Substances and Disease Registry (ATSDR) and the Environmental Protection Agency (EPA). The original guidelines were published in the *Federal Register* on April 17, 1987. Each profile will be revised and republished as necessary.

The ATSDR toxicological profile succinctly characterizes the toxicologic and adverse health effects information for the hazardous substance described therein. Each peer-reviewed profile identifies and reviews the key literature that describes a hazardous substance's toxicologic properties. Other pertinent literature is also presented, but is described in less detail than the key studies. The profile is not intended to be an exhaustive document; however, more comprehensive sources of specialty information are referenced.

The focus of the profiles is on health and toxicologic information; therefore, each toxicological profile begins with a public health statement that describes, in nontechnical language, a substance's relevant toxicological properties. Following the public health statement is information concerning levels of significant human exposure and, where known, significant health effects. The adequacy of information to determine a substance's health effects is described in a health effects summary. Data needs that are of significance to protection of public health are identified by ATSDR and EPA.

Each profile includes the following:

(A) The examination, summary, and interpretation of available toxicologic information and epidemiologic evaluations on a hazardous substance to ascertain the levels of significant human exposure for the substance and the associated acute, subacute, and chronic health effects;

(B) A determination of whether adequate information on the health effects of each substance is available or in the process of development to determine levels of exposure that present a significant risk to human health of acute, subacute, and chronic health effects; and

(C) Where appropriate, identification of toxicologic testing needed to identify the types or levels of exposure that may present significant risk of adverse health effects in humans.

The principal audiences for the toxicological profiles are health professionals at the Federal, State, and local levels; interested private sector organizations and groups; and members of the public. We plan to revise these documents in response to public comments and as additional data become available. Therefore, we encourage comments that will make the toxicological profile series of the greatest use.

Comments should be sent to:

Agency for Toxic Substances and Disease Registry
Division of Toxicology
Mail Stop E-29
Atlanta, Georgia 30333

The toxicological profiles are developed in response to the Superfund Amendments and Reauthorization Act (SARA) of 1986 (Public Law 99-499) which amended the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA or Superfund). This public law directed ATSDR to prepare toxicological profiles for hazardous substances most commonly found at facilities on the CERCLA National Priorities List and that pose the most significant potential threat to human health, as determined by ATSDR and the EPA. The availability of the revised priority list of 275 hazardous substances was announced in the *Federal Register* on April 29, 1996 (61 FR 18744). For prior versions of the list of substances, see *Federal Register* notices dated April 17, 1987 (52 FR 12866); October 20, 1988 (53 FR 41280); October 26, 1989 (54 FR 43619); October 17,1990 (55 FR 42067); October 17, 1991 (56 FR 52166); October 28, 1992 (57 FR 48801); and February 28, 1994 (59 FR 9486). Section 104(i)(3) of CERCLA, as amended, directs the Administrator of ATSDR to prepare a toxicological profile for each substance on the list.

This profile reflects ATSDR's assessment of all relevant toxicologic testing and information that has been peer-reviewed. Staff of the Centers for Disease Control and Prevention and other Federal scientists have also reviewed the profile. In addition, this profile has been peer-reviewed by a nongovernmental panel and is being made available for public review. Final responsibility for the contents and views expressed in this toxicological profile resides with ATSDR.

David Satcher, M.D., Ph.D
Administrator
Agency for Toxic Substances and
Disease Registry

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1. PUBLIC HEALTH STATEMENT

This public health statement tells you about lead and the effects of exposure.

The Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are the sites targeted for long-term federal cleanup. Lead has been found in at least 1,026 of the 1,445 current or former NPL sites. However, it's unknown how many NPL sites have been evaluated for this substance. As more sites are evaluated, the sites with lead may increase. This is important because exposure to this substance may harm you and because these sites may be sources of exposure.

When a substance is released from a large area, such as an industrial plant, or from a container, such as a drum or bottle, it enters the environment. This release does not always lead to exposure. You are exposed to a substance only when you come in contact with it. You may be exposed by breathing, eating, or drinking the substance or by skin contact.

If you are exposed to lead, many factors determine whether you'll be harmed. These factors include the dose (how much), the duration (how long), and how you come in contact with it. You must also consider the other chemicals you're exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

1.1 WHAT IS LEAD?

Lead is a naturally occurring bluish-gray metal found in small amounts in the earth's crust. It has no characteristic taste or smell. Metallic lead does not dissolve in water and does not burn. Lead can combine with other chemicals to form what are usually known as lead salts. Some lead salts dissolve in water better than others. Some natural and manufactured substances contain lead but do not look like lead in its metallic form. Some of these substances can burn—for example, organic lead compounds in some gasolines.

Table 2-1. Health Effects Associated with Exposure to Lead and Internal Lead Doses in Humans

Duration of exposure	System	Effect	Blood lead levels at which effect was observed (µg/dL)	Reference
>1 year (occup)		Increase in death due to hypertension, nephritis, neoplasms	63–80	Cooper et al. 1985, 1988
NS (occup)		Increase in death due to cerebrovascular disease, nephritis, and/or nephrosis	NS	Fanning 1988; Malcolm and Barnett 1982; Michaels et al. 1991
>3 years (occup)		No increase in deaths	34–58 (means)	Gerhardsson et al. 1986b
NS		Acute encephalopathy resulting in death in children	125–750	NAS 1972
2 weeks to >1 year (occup)	Cardiovascular	Increased blood pressure	≥30–120	deKort et al. 1987; Pollock and Ibels 1986; Marino et al. 1989; Weiss et al. 1986, 1988
>1 year (occup)	Cardiovascular	No effect on blood pressure	40 (mean)	Parkinson et al. 1987
>1 year (occup)	Cardiovascular	Ischemic electrocardiogram changes	51 (mean)	Kirkby and Gyntelberg 1985
NS (general population)	Cardiovascular	Increased blood pressure	44.9 (mean)	Khera et al. 1980b
NS (general population)	Cardiovascular	Increased systolic pressure by 1–2 mm Hg and increased diastolic pressure by 1.4 mm Hg with every doubling in blood lead level; effect most prominent in middle-aged white men	7–38	Coate and Fowles 1989; Harlan 1988; Harlan et al. 1988; Landis and Flegal 1988; Pirkle et al. 1985; Schwartz 1988
NS (general population)	Cardiovascular	No significant correlation between blood pressure and blood lead levels	6–13 (median) or NS	Elwood et al. 1988; Grandjean et al. 1989; Neri et al. 1988; Staessen et al. 1990, 1991
NS (general population)	Cardiovascular	Degenerative changes in myocardium, electrocardiogram abnormalities in children	6–20	Silver and Rodriquez-Torres 1968
NS (acute) (general population)	Gastrointestinal	Colic in children	60–100	EPA 1986a; NAS 1972

Table 2-1. Health Effects Associated with Exposure to Lead and Internal Lead Doses in Humans (continued)

Duration of exposure	System	Effect	Blood lead levels at which effect was observed (µg/dL)	Reference
NS (acute) (occup)	Gastrointestinal	Colic (abdominal pain, constipation, cramps, nausea, vomiting, anorexia, weight loss)	400–200	Awad et al. 1986; Baker et al. 1979; Haenninen et al. 1979; Holness and Nethercott 1988; Kumar et al. 1987; Marino et al. 1989; Matte et al. 1989; Muijser et al. 1987; Pagliuca et al. 1990; Pollock and Ibels 1986; Schneitzer et al. 1990
NS (occup)	Hematological	Increased ALAS and/or decreased ALAD	87 or NS (correlation with blood lead level)	Alessio et al. 1976; Meredith et al. 1978; Wada et al. 1973
NS (general population)	Hematological	Decreased ALAD	3–56 (adult) No threshold (children)	Chisholm et al. 1985; Hernberg and Nikkanen 1970; Lauwerys et al. 1978; Roels and Lauwerys 1987; Roels et al. 1976; Secchi et al. 1974
NS (occup)	Hematological	Increased urinary or blood ALA	>35 (adult) 25–75 (children)	Lauwerys et al. 1974; Meredith et al. 1978; Pollock and Ibels 1986; Selander and Cramer 1970; Solliway et al. 1996
NS (general population)	Hematological	Increased urinary ALA	>35 (adult) 25–75 (children)	NAS 1972; Roels and Lauwerys 1987
NS (general population)	Hematological	Increased FEP	≥25–35	Grandjean and Lintrup 1978; Roels et al. 1975
NS (general population)	Hematological	Increased EP	30–40 (males) 20–30 (females)	Roels and Lauwerys 1987; Roels et al. 1975, 1976, 1979; Stuick 1974
NS (general population)	Hematological	Increased ZPP	≥15 (children)	Hammond et al. 1985; Piomelli et al. 1982; Rabinowitz et al. 1986; Roels and Lauwerys 1987; Roels et al. 1976
1-28 years (occup)	Hematological	Increased ZPP and urinary ALA	51 (mean) 40–75 (range)	Gennart et al. 1992a
NS (general population)	Hematological	Increased urinary coproporphyrin	≥35 (children ≥40 (adults)	EPA 1986a
NS (general population)	Hematological	Decreased hemoglobin	≥40 (children)	Adebonojo 1974; Betts et al. 1973; Pueschel et al. 1972; Rosen et al. 1974

Table 2-1. Health Effects Associated with Exposure to Lead and Internal Lead Doses in Humans (continued)

Duration of exposure	System	Effect	Blood lead levels at which effect was observed (µg/dL)	Reference
NS (occup)	Hematological	Decreased hemoglobin with or without basophilic stippling of erythrocytes	≥40	Awad et al. 1986; Baker et al. 1979; Grandjean 1979; Lilis et al. 1978; Pagliuca et al. 1990; Tola et al. 1973; Wada et al. 1973
NS (general population)	Hematological	Anemia (hematocrit of <35%)	>20 (children)	Schwartz et al. 1990
NS (occup)	Hematological	Decreased Py-5'-N	NS	Buc and Kaplan 1978; Paglia et al. 1975, 1977
NS (general population)	Hematological	Decreased Py-5'-N	7–80 (children)	Angle and McIntire 1978; Angle et al. 1982
NS (acute) (general population)	Hepatic	Decreased mixed function oxidase activity	NS (children)	Alvares et al. 1975; Saenger et al. 1984
NS (chronic) (occup)	Renal	Chronic nephropathy	40–>100	Biagini et al. 1977; Cramer et al. 1974; Lilis et al. 1968; Maranelli and Apostoli 1987; Ong et al. 1987; Pollock and Ibels 1986; Verschoor et al. 1987; Wedeen et al. 1979
1–30 years (occup)	Renal	No effect on renal function	40–75	Buchet et al. 1980; Huang et al. 1988a; Gennart et al. 1992a
NS (chronic) (general population)	Renal	Renal impairment with gout or hypertension	18–26	Batumen et al. 1981, 1983
NS (acute) (general population)	Renal	Aminoaciduria; Fanconi syndrome	>80 (children)	Chisolm 1962; Pueschel et al. 1972
0.2–20 years (chronic) (occup)	Endocrine	Decreased thyroxin (T_4)	≥56	Tuppurainen et al. 1988
1–28 years (occup)	Endocrine	No effect on thyroid hormones, TSH, LH, and FSH	51 (mean) 40–75 (range)	Gennart et al. 1992a

Table 2-1. Health Effects Associated with Exposure to Lead and Internal Lead Doses in Humans (continued)

Duration of exposure	System	Effect	Blood lead levels at which effect was observed (µg/dL)	Reference
NS (chronic) (general population)	Endocrine	No effect on thyroid function in children	2–77 (levels measured)	Siegel et al. 1989; Huseman et al. 1992
NS (general population)	Other	Negative correlation between blood lead and serum 1,25-dihydroxyvitamin D in children	12–120	Mahaffey et al. 1982; Rosen et al. 1980
NS (chronic) (general population)	Other	No effect on vitamin D metabolism in children	5–24 (levels measured)	Koo et al. 1991
NC (chronic) (general population)	Other	Growth retardation in children	≥30–60; Tooth lead >18.7 µg/g	Angle and Kuntzelman 1989; Lauwers et al. 1986; Lyngbye et al. 1987; Huseman et al. 1992
NS (chronic) (general population)	Other	No association between blood lead levels and growth in children	10–47 (levels measured)	Greene and Ernhart 1991; Sachs and Moel 1989
NS (general population)	Other	Decreased growth rate	7.7	Shukla et al. 1989, 1991
NS (Mexican-American children)	Other	Decreased stature	≥9–10	Frisancho and Ryan 1991
<18 years (occup)	Immunological	Depression of cellular immune function, but no effect on humoral immune function	21–90	Alomran and Shleamoon 1988; Ewers et al. 1982
NS (acute)	Neurological	Encephalopathy (adults)	50–>300	Kehoe 1961a; Kumar et al. 1987; Smith et al. 1938
NS (occup)	Neurological	No effect on neurobehavioral function in adults	40–60 (levels measured)	Milburn et al. 1976; Ryan et al. 1987
NS (occup)	Neurological	No effect on peripheral nerve function	60–80 (levels measured)	Spivey et al. 1980

Table 2-1. Health Effects Associated with Exposure to Lead and Internal Lead Doses in Humans (continued)

Duration of exposure	System	Effect	Blood lead levels at which effect was observed (µg/dL)	Reference
NS (acute and chronic) (occup)	Neurological	Neurological signs and symptoms in adults including malaise, forgetfulness, irritability, lethargy, headache, fatigue, impotence, decreased libido, dizziness, weakness, paresthesia	40–80	Awad et al. 1986; Baker et al. 1979; Campara et al. 1984; Haenninen et al. 1979; Holness and Nethercott 1988; Marino et al. 1989; Matte et al. 1989; Pagliuca et al. 19990; Parkinson et al. 1986; Pasternak et al. 1989; Pollock and Ibels 1986; Schneitzer et al. 1990; Zimmerman-Tansella et al. 1983
NS (occup)	Neurological	Neurobehavioral function in adults; disturbances in oculo-motor function, reaction time, visual motor performance, hand dexterity, IQ test and cognitive performance, nervousness, mood, coping ability, memory	40–80	Arnvig et al. 1980; Baker et al. 1983; Baloh et al. 1979; Campara et al. 1984; Glickman et al. 1984; Haenninen et al. 1978; Hogstedt et al. 1983; Mantere et al. 1982; Maizlish et al. 1995; Spivey et al. 1980; Stollery et al. 1989, 1991; Valciukas et al. 1978; Williamson and Teo 1986
NS (occup)	Neurological	Peripheral nerve function in adults; decreased nerve conduction velocity	30–≥70	Araki et al. 1980; Muijser et al. 1987; Rosen et al. 1983; Seppalainen et al. 1983; Triebig et al. 1984
NS (general population)	Neurological	Irritability, lethargy, behavioral problems and encephalopathy in children	60–450 (effects other than encephalopathy); >80–800 (encephalopathy)	Bradley and Baumgartner 1958; Bradley et al. 1956; Chisolm 1962, 1965; Chisolm and Harrison 1956; Gant 1938; Rummo et al. 1977; Smith et al. 1983
NS (general population)	Neurological	Neurobehavioral function in children: slightly decreased performance on IQ tests and other measures of neuropsychological deficits	40–200	dela Burde and Choate 1972, 1975; Ernhart et al. 1981; Kotok 1972; Kotok et al. 1977; Rummo et al. 1979
NS (general population)	Neurological	Neurobehavioral function in children: slightly decreased performance on IQ tests and other measures of neuropsychological function	Tooth lead: 60–>30 µg/g Blood lead: 6–60	Bellinger and Needleman 1983; Bergomi et al. 1989; Fulton et al. 1987; Hansen et al. 1989; Hawk et al. 1986; Needleman et al. 1979, 1985, 1990; Schroeder and Hawk 1987; Schroeder et al. 1985; Silva et al. 1988; Wang et al. 1989

Table 2-1. Health Effects Associated with Exposure to Lead and Internal Lead Doses in Humans (continued)

Duration of exposure	System	Effect	Blood lead levels at which effect was observed (µg/dL)	Reference
NS (general population)	Neurological	No correlation between blood lead levels and permanent effects on neurobehavioral development in children	10–15	Bellinger et al. 1989a; Cooney et al. 1989a; Dietrich et al. 1987a; Ernhart et al. 1990; Harvey et al. 1984, 1988; Lansdown et al. 1986; McBride et al. 1982; McMichael et al. 1986; Pocock et al. 1989; Smith et al. 1983; Winneke et al. 1984
NS (general population)	Neurological	Altered auditory evoked potential latency and decreased hearing acuity in children	4–60	Holdstein et al. 1986; Robinson et al. 1985; Schwartz and Otto 1987
NS (general population)	Neurological	Postural disequilibrium	11.9 geometric mean for first 5 years of age	Bhattacharya et al. 1993
NS (general population)	Neurological	Peripheral neuropathy and reduced conduction velocity in children	20–30	Erenberg et al. 1974; Landringan et al. 1976; Schwartz et al. 1988; Seto and Freeman 1964
Prenatal (general population)	Developmental	Reduced birth weight and/or reduced gestational age, and/or increased incidence of stillbirth and neonatal death	12–17	Bornschein et al. 1989; McMichael et al. 1986; Moore et al. 1982; Wart et al. 1987; Wibberley et al. 1977
NS (general population)	Developmental	No association between blood lead levels and birth weight, gestational age, or other neonatal size measures	3–55	Greene and Ernhart 1991; Factor-Litvak et al. 1991
NS (general population)	Developmental	Impaired mental development in children	10–15	Baghurst et al. 1987; Bellinger et al. 1984, 1985a, 1985b, 1986a, 1986b, 1987a, 1987b; Bornschein et al. 1989; Deitrich et al. 1986, 1987a, 1987b; Earnhart et al. 1985, 1986, 1987; McMichael et al. 1988; Rothenberg et al. 1989a; Wigg et al. 1988; Winneke et al. 1985a, 1985b; Wolf et al. 1985; Vimpani et al. 1985, 1989

Table 2-1. Health Effects Associated with Exposure to Lead and Internal Lead Doses in Humans (continued)

Duration of exposure	System	Effect	Blood lead levels at which effect was observed (µg/dL)	Reference
NS (general population)	Developmental	Impaired motor developmental status in 6-year old children (Cincinnati cohort)	≥9.0 (mean lifetime)	Dietrich et al. 1993b
NS (general population)	Developmental	Moderate deficit in Wechsler Performance IQ in children 6.5 years old (Cincinnati cohort)	≥20 (average lifetime)	Dietrich et al. 1993a
NS (general population)	Developmental	Lower scores in test of Cognitive Function at 5 and 10 years of age	6.5 (mean at 24 months of age)	Bellinger et al. 1991, 1992
NS (general population)	Developmental	Inverse correlation between blood lead levels and ALA and ALAD activity	10–33 (mean)	Haas et al. 1972; Kuhnent et al. 1977; Lauwerys et al. 1978
NS (occup)	Reproductive	Decreased fertility	37.2 (mean)	Lin et al. 1996
NS (general population)	Reproductive	Increased incidence of miscarriages and stillbirths in exposed women	≥10 or NS	Baghurst et al. 1987; Hu et al. 1991; McMichael et al. 1986; Nordstrom et al. 1979; Wibberley et al. 1977
NS (general population	Reproductive	No association between blood lead levels and the incidence of spontaneous abortion in exposed women	2	Murphy et al. 1990
NC (occup)	Reproductive	Low sperm count, decreased sperm mobility, abnormal sperm	40–50	Alexander et al. 1996; Assennato et al. 1987; Braunstein et al. 1978; Chowdhury et al. 1986; Cullen et al. 1984; Lancranjan et al. 1975; Rodamilans et al. 1988; Wildt et al. 1983

ALA = δ-aminolevulinic acid; ALAD = δ-aminolevulinic acid dehydratase; ALAS = δ-aminolevulinic acid synthase; EP = erythrocyte protoporphyrins; FEP = free erythrocyte protoporphyrins; FSH = follicule stimulating hormone; IQ = intelligence quotient; LH = luteneizing hormone; NS = not specified; (occup) = occupational; Py-5'-N = pyrimidine-5-nucleotidase; TSH = thyroid stimulating hormone; ZPP = erythrocyte protoporphyrin

Human Health
Technical Committee
Reports

DRAFT

Please address comments on this draft report to: ¥ ACERP (public comment)
¥ Arizona Department of Environemtal Quality ¥ 3033 North Central Avenue
¥ Phoenix, Arizona 85012-2809

9. LEAD
Will Humble

HEALTH EFFECTS OF LEAD
Lead (also known as lead metal, plumbum, and pigment metal) is an element found throughout the environment in the earth's crust and from processes initiated by humans. It is found in air, food, water, and dust.
The routes of exposure for lead include inhalation, ingestion, or dermal contact. If deposition of lead particles occurs in the lower respiratory tract, the particle absorption is almost total. Fifty percent of the lead which is ingested by children is absorbed by the body with an 8% and 15% rate of absorption in two separate studies examining ingestion exposure in adults. Fasting has been shown to enhance ingestion absorption to up 45% in adults. In animals, the absorption of alkyl lead (tetraethyl lead) occurred more rapidly by dermal application in rabbits than by ingestion. Since human's dermal absorption rate is lower, absorption in humans by dermal contact is less than by inhalation or ingestion. Ingestion is the most frequent exposure route for children. Inorganic lead is not metabolized or biotransformed; however, metabolism does occur in the liver with organic (alkyl) lead. Regardless of the route of absorption, lead is distributed in the blood, soft tissue, and bone with the majority of the total body burden in the bone. Excretion in humans occurs through the urine and feces. Transplacental transfer has also been observed in humans.
A variety of toxic effects have been documented in humans from inhalation and ingestion exposures to lead. Severity of symptoms is dose dependent with higher doses of lead producing more severe symptoms. Impairment of heme synthesis with resultant anemia has been seen. Neurobehavioral toxicity has been documented in occupational groups mainly from inhalation but also from ingestion. Lead encephalopathy is the most serious neurobehavioral effect with symptoms of dullness, irritability, poor attention span, headache, muscular tremor, memory loss, and hallucinations. If the exposure concentration is high enough, the condition becomes quite severe, resulting in coma and death. Acute encephalopathy and death have been documented in children with mainly ingestion and secondarily inhalation exposure. At lower lead concentration levels, children have manifested neurological impairment (hyperactivity, peripheral neuropathy) and cognitive deficits (lower IQ). With inhalation and ingestion, some of the other consequences of lead exposure include cardiovascular toxicity (abnormal EKGs, high blood pressure), nephropathy, interference with Vitamin D metabolism, gastrointestinal symptoms (colic), developmental toxicity (low birth weight), growth retardation, compromise of the immune system, and reproductive toxicity (miscarriage). Studies in these areas for dermal exposures were not available.
Data in epidemiologic studies were not adequate to establish an association between lead exposure and the development of cancer. Failure to document the specific lead compound, its dose, and the compound's exposure routes were all weaknesses of these studies. An examination of lead production and battery workers who had inhaled lead in the workplace has demonstrated higher rates of total malignancies and mortality from total malignancies than would otherwise have been expected. For example, an increased number of renal cancers was observed in lead smelter workers. In a number of animal studies, kidney tumors have consistently been reported with lead ingestion exposure. Lead has an EPA classification of B2, probable human carcinogen (USDHHS, 1992). The ADHS is the state agency mandated by Arizona law to maintain a registry for recording cases with elevated blood lead levels. In March, 1993, the Arizona lead poisoning reporting law was revised. The reporting level was changed from 25 microns/dL to 10 microns/dL.

ENVIRONMENTAL EXPOSURES TO LEAD

ENVIRONMENTAL EXPOSURES TO LEAD

Introduction

Exposure to environmental media containing lead is the primary source of elevated blood lead in children. Sources of lead which may contribute to total lead exposure include water, air, soil, and dust. Lead based paint, ingested as paint chips or dust is the most common high dose source of lead nationwide, and is the source most commonly identified in Arizona cases. Other sources may include food, ceramic and pewter cooking utensils, home projects such as ceramics-making, and home remedies that contain lead.

Quantifying lead exposures from all sources is a critical part of estimating blood lead levels in children. This section analyzes and discusses the relative contributions made by each of these lead sources. Information about relative contributions may be used to effectively direct intervention resources.

We used two methods to assess environmental exposure to lead. The first method uses the EPA Integrated Exposure Uptake Biokinetic (IEUBK) model to evaluate lead exposure (USEPA, 1994). The second method uses epidemiological data collected by the lead exposure registry at the ADHS as a measure of elevated blood lead levels in the population.

The IEUBK model generates a probability distribution of blood lead levels for a group of children exposed to a particular concentration of lead in each media. The distribution reflects the variability of blood lead levels in several communities.

Methods

The IEUBK model was run four times using representative input parameters for environmental concentrations of lead which contribute to total lead exposure. The populations for which analyses were conducted include:

¡ Children living in areas with low background levels of soil lead whose homes do not contain lead based paint.

¡ Children living in areas with low background levels of soil lead whose homes contain lead based paint.

¡ Children living in areas with higher background levels of soil lead (i.e., smelter towns) whose homes do not contain lead based paint.

¡ Children living in areas with higher background levels of soil lead whose homes contain lead based paint.

The following paragraphs discuss each of the exposure media that may contribute to total lead exposure. IEUBK input parameters for each media are also discussed.

Water

Data from a computerized data base from the ADEQ were used to estimate lead exposures from water in Arizona and its contribution to total lead exposure. Data were available for 72% of the service population in Arizona. About 95% of the analyzed population in Arizona are being exposed to less than 0.0099 mg/L, and 99.2% are exposed to less than 0.0199 mg/L. A subpopulation of approximately 7000 persons are being served by water systems with lead in excess of 0.06 mg/L. The IEUBK model was run using a concentration of 0.0099 mg/L as an estimate of statewide lead exposure from water. This estimate is conservative and will tend to overestimate the contribution of lead exposure via water.

Air

Data from an ADEQ publication (1992) were used to estimate lead exposures from outdoor air in Arizona and its contribution to total lead exposure. Data collected from the Phoenix and Tucson

metropolitan were used in the analysis as these data represent the majority of the population in Arizona. Data from the Chiricahua National Monument was used in the Douglas/Bisbee analysis. A total of 263 sample analyses were available for Phoenix and Tucson, and 93 were available for Chiricahua National Monument. All data represented lead particles in the respirable fraction (<10 microns). The mean of four quarterly averages from 263 samples at five sample locations (0.83 micron/m3) was used as the urban exposure concentration. The mean of four quarterly averages from 93 samples at one location (4.5 microns/m3) was used in the Douglas/Bisbee analysis.
The IEUBK model was run using 0.83 micron/m3 as the estimated lead concentration in air in urban areas. A value of 4.5 microns/m3 was used for the Douglas/Bisbee analysis.

Soil
Data from the ADEQ (Earth Technology Inc., 1991) and a 1985 soil and blood lead investigation conducted by the ADHS in Douglas and Bisbee were used to evaluate lead exposures from soils. The ADEQ data includes 62 soil samples from eight locations in the Phoenix and Tucson areas. The Douglas/Bisbee data includes 126 soil samples from 126 locations. The urban and Douglas/Bisbee data were averaged in order to provide two estimates for input into the model.
The IEUBK model was run using the calculated mean of 7.7 mg/kg as the estimated lead concentration in soil in urban areas. A value of 303 mg/kg was used for the Douglas/Bisbee analysis.

House Dust
Lead in house dust is often usually composed of lead from soils and airborne fallout. Other sources may include lead based paint in the home. No data are available on the concentration of lead in house dust; however, an estimate can be made if soil and air concentrations are known. Since lead based paint in a home can significantly influence the amount of lead in the house dust, this analysis breaks lead exposure into homes with and without lead based paint. The IEUBK model uses a multiple source analysis to predict lead concentrations in house dust based upon the concentrations of lead in soil and air. Separate models were run for both the urban and Douglas/Bisbee assessment for homes with lead based paint. The IEUBK model was run using the soil lead concentrations discussed in the previous section as the concentration in house dust in homes without lead based paint. The concentration of lead in house dust from paint was assumed to be 1200 mg/kg in homes with lead based paint. The model integrates sources from soil and lead based paint to obtain an estimate of lead in house dust.

Diet
The contribution of total lead exposure from food was assessed using the default parameters included in the IEUBK model. Exposure to lead from foods in the model is based on an analysis of the typical American diet and quarterly surveys of lead amounts in this diet.

Results
Input parameters for each population group were determined using the data presented above. Modeling parameters and probability distributions are presented in Appendix A. The following paragraphs discuss the modeling results.
¡ Children living in areas with low background levels of lead in soil and whose houses do not contain lead based paint are unlikely to have elevated blood lead. The probability distribution for this population indicates that 0.06% of the children under six years old in this population will have blood lead concentrations in excess of 10 microns/dL. The geometric mean concentration of blood lead predicted by the model is 2.2 microns/dL.

¡ Some children living in areas with higher levels of lead in soil and whose houses do not contain lead based paint may have elevated blood lead. The probability distribution for this population indicates that about 13% of the children under six years old in this population will have blood lead concentrations greater than 10 microns/dL. The geometric mean concentration of blood lead predicted by the model is 6 microns/dL.

¡ Children living in areas with low background levels of lead in soil and whose houses contain lead based paint are more likely to have elevated blood lead. The probability distribution for this population indicates that about 34% of the children under six years old in this population will have blood lead concentrations in excess of 10 microns/dL. The geometric mean concentration of blood lead predicted by the model is 8.5 microns/Dl.

¡ Children living in areas with higher background levels of lead in soil (303 mg/kg) and whose houses contain lead based paint are the group most likely to have elevated blood lead. The probability distribution for this population indicates that about 50% of the children under six years old in this population will have blood lead concentrations in excess of 10 microns/dL. The geometric mean concentration of blood lead predicted by the model is about 10 microns/dL.

Model Estimate of Number of Children with Elevated Blood Lead
According to the IEUBK model, children living in homes that contain lead paint are at the highest risk of lead poisoning as measured by elevated blood lead. Children living in areas with higher levels of soil lead are at some risk for lead poisoning; however, the likelihood of elevated blood lead in this population is less than that of elevated blood lead in children in homes that contain lead paint. Children living in areas with low background levels of lead in soil and whose houses do not contain lead based paint are unlikely to have elevated blood lead.
Lead was widely used as a major ingredient in most oil based paints before the 1950s. In 1972, the Consumer Products safety Commission limited lead content in new paint to 5,000 mg/kg, and to 600 mg/kg in 1978. Due to the quantity of lead in paint before 1950, homes built before this time are the most likely to contain lead based paint. Ninety percent of homes built before 1940 contain some lead

based paint.
According to 1990 census data, approximately 3.2% of the housing units in Arizona were built before 1940 and 6.8% were built before 1950. The data indicates that about 93,000 persons are living in these pre-1950 houses in Arizona. Assuming that approximately 11% of this population are under the age of six (ADES, 1993), approximately 10,230 children aged 6 and less are in the highest risk category. The IEUBK model predicts that 34% of the 10,230 at-risk children, or 3,478 children, may have elevated blood lead in this category. Assuming that the remainder of the under six population is in the lowest risk group (460,200), and that 0.06% of these children will have elevated blood lead, an additional 282 cases are predicted. The total number of children with elevated blood lead estimated by the model is about 3,760. The IEUBK model considers all of the
environmental sources discussed in Section II, including air, water, food, soil and house dust. In addition, the model assumes an average maternal blood lead concentration of 2.5 microns/dL. Analysis of the model results indicates that the majority of total lead exposure is via ingestion of lead containing soil and house dust.

Modeling Uncertainties
"Because a child's exposure to lead involves a complex array of variables, because there is population sampling variability, and because there is variability in environmental lead measurements and background levels of lead in soil, dust, food and drinking water, results from the (IEUBK) model may differ from results of blood lead screening of children in a community." (USEPA, 1994b) In addition, possible sources of lead exposure that are not considered by the model include the use of ceramic or pewter cooking utensils, home projects such as ceramics-making, and the use of home remedies that contain lead.

ELEVATED BLOOD LEAD REPORTING AND
INVESTIGATIONS
National Trends
The concentrations of lead in the blood of children has declined substantially in recent years. A national survey of blood lead levels taken from 1988-1991 found that lead levels in children one through five years of age had decreased 88% since a survey conducted from 1976-1980 (USDHHS, 1994). The 1976-1980 study found that 53% of children aged one through five had blood lead levels over 15 microns/dL and 9.3% had levels over 25 microns/dL. The 1988-1991 study found that 2.7% of children aged one through five had blood lead levels over 15 microns/dL and 0.5% had levels over 25 microns/dL.

Much of the reduction in blood lead levels may be attributed to a 99.8% reduction in the amount of lead in gasoline and the virtual elimination of lead solder in food and soft-drink cans (USDHHS, 1994). Further reductions in blood lead levels will require elimination of deteriorated leaded paint in older housing, and a reduction of lead concentrations in contaminated soils.

Statewide Reporting and Investigations
The number of reported lead poisoning cases in Arizona rose dramatically in 1993. The ADHS lead registry received 315 cases of elevated blood lead levels in 1993, a 573% increase over the previous year. This increase is most likely due to the recent changes in reporting and screening (ADHS, 1993). The Arizona Health Care Cost Containment System began screening Arizona children through the Early Periodic Screening, Diagnosis, and Treatment program in 1993. However, no rates can be determined using these data since the total number of children that have been screened is not available.

The primary suspected source of lead exposure in children during 1993 was lead based paint. Other suspected sources included lead containing soil, ceramic and pewter cooking utensils, ceramicsmaking, and "take-home" occupational exposures. In addition, a number of cases may have been caused by water stored in containers with lead-containing solder and brass fittings.

The majority of cases were from Maricopa (62%) and Pima Counties (23%). The distribution of elevated blood lead cases may reflect the size of the local populations and the numbers of children screened in each county.

In 1993, there were 144 children in Douglas and Pirtleville screened for elevated blood lead by the Arizona Health Care Cost Containment System. The screening found three (2%) with blood lead concentrations in excess of 10 microns/dL. There have been seven reported cases of elevated blood lead in the Douglas area during 1994 (as of August 1, 1994). It is not known how many children were tested to yield these cases. Of the seven cases, four were sufficiently high to warrant environmental investigations. Paint and paintcontaminated soil were identified as the most likely sources for one case. The folk remedy Azarcon was implicated in three cases.

Epidemiological Estimate of Number of Children with Elevated Blood Lead.
While there is no published estimate of the rate of elevated blood lead levels in Arizona, estimates of the number of potential cases can be made by applying rates found in clinical settings to the number of children in Arizona.

The 1988-1991 NHANES study estimated that 2.7% of children nationally may have lead levels in excess of 15 microns/dL, with 0.5% of children having levels in excess of 25 microns/dL. The number of children under age six in Arizona in 1994 is estimated to be 470,430 (ADES, 1993).

Data collected by the University of Arizona Medical Center Pediatric Clinic found that 2.3% of 1,439 children screened for blood lead from March 1993 through January, 1995 had blood lead levels between 10 microns/dL and 14 microns/dL; 0.3% had levels between 15 and 19 microns/dL; and 0.1% had levels between 20 and 24 microns/dL (University of Arizona, 1995). The age distribution of the children screened is not provided in the memorandum; however, the majority of children screened were probably less than three years old.

The estimate of the number of children in Arizona with elevated blood lead made here uses the percentages from the University of Arizona memorandum as a measure of the percentage of children with elevated blood lead in Arizona. If one assumes that the percentages found at the University of Arizona are representative of all children ages 0-6 years in Arizona, then the number of children with blood lead levels in excess of 10 microns/dL in Arizona may be 12,700, with 470 of these in excess of 20 microns/dL. However, the vast majority of reported elevated blood lead cases to the ADHS registry are less than three years of age, and the majority of screening occurs in this population. Additionally, the vast majority of the screening data from the University of Arizona is in the 0-3 age group. If one assumes that the percentages found at the University of Arizona are representative of children aged 0-3 years, then the number of children with blood lead levels in excess of 10 microns/dL in Arizona may be 6,350 with 235 of these in excess of 20 microns/dL.

CONCLUSION
Children with elevated blood lead levels are at risk of developing neurological damage, including peripheral neuropathy, hyperactivity, and cognitive deficits (decreased IQ).

The estimated number of children in Arizona with blood lead levels in excess of 10 microns/dL is 6,350. An estimated 235 of these children may have severely elevated blood lead (>20 microns/dL).

Considerable progress has been made in reducing childhood exposure to lead through the phase-out of leaded gasoline, lead-soldered cans, and lead based paint. However, residual lead contamination in houses with old lead based paint remains in some areas in Arizona. While there are a number of media through which children may be exposed to lead, eliminating exposure to lead based paint and lead contaminated soils will substantially reduce lead exposure in children.

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A.1.3

The Philadelphia Inquirer, December 23, 1998

Junkyard operator told to pay

A federal judge said he owed the government $8.7 million for cleanup near Honey Brook.

By Paul Nussbaum
INQUIRER STAFF WRITER

A federal district judge has ordered a Chester County junkyard operator to pay about $8.7 million to state and federal agencies for cleanup costs at an old landfill that contaminated nearby groundwater.

Senior U.S. District Judge Clifford Scott Green on Friday ordered Ernest Barkman, his wife, Grace, and three corporations owned by Barkman to pay the millions to reimburse the federal Environmental Protection Agency and the state Department of Environmental Protection for testing for contamination, supplying 44 local residents with bottled water, and then connecting their homes to a municipal water system. The judge also ordered the Barkmans to pay $184,500 in fines for disobeying an EPA order to vacate the site by Nov. 28, 1993.

Barkman and his wife ran a landfill near Honey Brook Township in western Chester County from 1963 until 1977. Since then, they have operated a truck repair and metal salvage yard at the Welsh Road location. The property was declared a Superfund site in 1984 after testing revealed arsenic, lead, cadmium, mercury, benzene, chloroform, and other hazardous substances in nearby groundwater.

A long-running battle to clean up the site ensued. Assistant U.S. Attorney Kier Dougall said yesterday the summary judgment against the Barkmans would allow the site to be cleared and a cap to be built over the former landfill to reduce health risks and future groundwater contamination.

Barkman declined comment yesterday, but his attorney, Francis X. Clark, said the Barkmans do not have the millions sought by the government. Clark said the Barkmans contend they are not responsible for the water contamination, and consider themselves pawns in a local effort to get rid of an unattractive business and replace it with residential development.

Clark said the Barkmans had not decided whether to appeal the court's decision, but "it's a little crazy to expect a little trash guy to pay for the community's water system."

1986-1997 Daily Southtown

State moves on auto dump

Midwest Metallics suit goes forward

Tuesday, August 12, 1997

By Rah Bickley
Staff Writer

For years, Summit village officials have threatened, cajoled and fretted as they watched a dump containing hazardous waste grow into a mountain four stories high and two football fields long.

They listened to explanations and excuses from the company dumping the debris and watched it break promise after promise.

Now the Illinois attorney general is stepping in to try to do what Summit could not do on its own: force the village's most notorious business to clean up its act.

Midwest Metallics, 7955 W. 59th St., a scrap metal recycler, was sued by the Illinois attorney general's office in October 1995 for having an illegal hazardous waste dump.

Until last week, the state was trying to settle the lawsuit by getting the company to remove the debris.

But when Midwest Metallics missed its third deadline this year to file a cleanup plan, the state stopped negotiating.

"We're going to proceed with litigation," attorney general spokesman Charles Jolie said.

Midwest Metallic crushes junked cars and sells the metal as scrap. The leftovers — such as car seats, dashboards and plastic bumpers — are shredded and turned into "auto fluff" that is piling up on the property.

The auto fluff is considered hazardous waste because it contains materials such as lead, cadmium and polychlorinated biphenyls, Jolie said. The attorney general's office estimates the dump's size at 700,000 to 800,000 cubic yards.

Lead and cadmium can damage the nervous system. PCBs can cause cancer and disrupt human endocrine systems, resulting in reproductive disorders, said Jack Darin of the

Sierra Club's Illinois chapter.

The waste pile could pose a public health threat if it caught fire and spewed toxic chemicals into the air, as happened in 1993 at a similar operation in Gary, or if heavy rains leached the chemicals into the ground or swept them into storm drains, state officials said.

In its lawsuit, the state contends that Midwest Metallics is violating water pollution and record-keeping requirements, illegally storing waste and creating a public nuisance. The lawsuit asks that the company be forced to remove all the fluff and take steps to restore the groundwater and soil as necessary, or to treat the site as a landfill with the appropriate pollution-control and record-keeping measures.

If the company loses the lawsuit, it faces a maximum fine of $400,000, plus possible daily fines for non-compliance.

On May 15, Midwest Metallics missed the attorney general's deadline to submit a cleanup plan. The company promised to submit the plan during the week of June 2, but still has not done so.

Phone calls on Friday and Monday to Midwest Metallics spokesman Terence Coogan and to Jim Barry, president of its parent company, National Materials in Elk Grove Village, were not returned.

The dump has been there for more than 30 years, Summit Trustee Cyril Lambert said. And Lambert believes it is growing. A new overnight truck shift has recently started hauling materials in and out of the plant, he said.

"We feel it's from the South Side," Lambert said. "I think that they're cleaning it up there and bringing it over here."

Midwest Metallics has operated plants in Summit, McCook, Joliet and the South Deering neighborhood of Chicago.

"I've been here 30 years. It's never changed. It's getting worse," Lambert said.

Summit officials have repeatedly threatened to revoke the company's business license if it doesn't move to clean up the site, but fear that doing so might cause Midwest Metallics to abandon the site without a cleanup. If that happened, Mayor Joseph Strzelczyk suggested, the company's principals might be personally liable.

Two defendants in the attorney general's lawsuit are named as individuals: Michael Tang, president of S.D. Metals Inc., a corporate partner of Midwest Metallics; and James Pielet, president of Pielet/Tang Enterprises Inc. The other defendants are Midwest Metallics; its predecessor, Pielet Bros. Scrap Iron & Metal Limited Partnership; S.D. Metals, and other companies.

Midwest Metallics bought the company from Pielet Bros. in 1993.

In addition to its alleged environmental violations, Midwest Metallics has been cited for numerous safety violations:

- Two workers died in January 1994 from toxic fumes while trying to drain an underground pipe. The U.S. Occupational Safety and Health Administration fined Midwest Metallics $61,000 and cited it for 22 safety violations it said posed serious risk of death or severe injury, including lack of protective equipment or worker safety training.

- Five months later, in June 1994, another worker drowned in the company wastewater pool. OSHA fined the company another $11,600 for three violations, including the lack of a guardrail around the pool.

- In December 1994, a worker at the company's Joliet plant was crushed to death between a truck and a forklift, which was later found to have no brakes. Six months later the company was assessed a $1 million fine, the largest ever issued to a Chicago-area scrap metal company. OSHA noted at the time that Midwest Metallics had inherited many of the hazards from the previous owners.

- In June 1995, OSHA cited the company for exposing workers to hazardous levels of lead, cadmium and arsenic.

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PACIFIC NORTHWEST POLLUTION PREVENTION RESEARCH CENTER

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Project Title: Automobile Shredder Residue - Recovering the Plastics in Junked Cars

Date Last Updated: 1/96

Project Summary:

Obsolete automobiles are the most recycled high-volume consumer product in the US. Currently, 92% of ferrous auto scrap is recovered for use as feedstock for the steel industry. However, for each ton of steel that is recovered, between 500-700 pounds of automobile shredder residue (ASR) is produced. ASR contains plastics, rubber, wood, paper, fabrics, glass, sand, dirt, metal pieces, and possibly automotive fluids and refrigerants; the current disposal technology is landfilling. When ASR is landfilled, many high-priced materials with significant embodied energy are lost. This loss, combined with the cost of landfilling, more stringent regulations, long-term viability concerns, and shrinking landfill space require new disposal alternatives for ASR.

This project will examine a process for recycling many of the plastics found in ASR. These plastics have potential use in foam rebonding and in the production of plastic blends and plastic parts. Also recovered during the process are iron- and silica-rich fines, whose potential for use as a fuel in pulverized coal combustion systems is being evaluated. The recycling process will yield a net energy savings of about 52 trillion Btu/year in 2010. Because the process recovers virtually all of the constituents of the ASR waste stream, there is a large reduction in the amount of solid waste that would otherwise be landfilled. Recovering the recyclable plastics will provide a new source of low-cost raw materials

for industry, reducing the need for imported petroleum feedstocks by millions of barrels of oil equivalent each year. The process will also directly reduce the import of as much as 100,000 tons/year of scrap industrial polyurethane foam, valued at $60 million, for the domestic rebonding industry.

Project Keywords: automobile shredder residue, plastics, recycling

Organization Performing Research: Argonne National Laboratory

Primary Research Contact:
Edward J. Daniels or Bassam J. Jody
Argonne National Laboratory
Building 362
9700 South Cass Ave.
Argonne, IL 60439
Daniels: (708) 252-5279
Jody: (708) 252-4206

Date Research Started/Completed: /92 ??? - /98

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Approximate Project Budget: $30,917,000

Primary Project Funder: DOE/EE/OIT/Industrial Waste Program

Funder Contact:

Simon "Sig" Friedrich

U.S. Department of Energy, EE-20

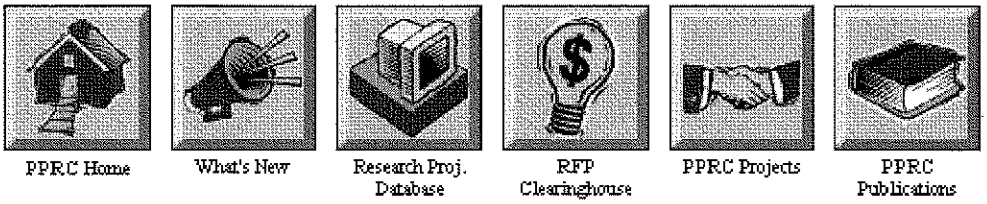
1000 Independence Ave., SW

Washington, DC 20585

(202) 586-6759

fax: (202) 586-8134

Created by the Pacific Northwest Pollution Prevention Research Center, 1326 Fifth Ave, Suite 650, Seattle, WA 98101
phone: (206) 223-1151, fax: (206) 223-1165, e-mail: office@pprc.org, WWW address: http://pprc.pnl.gov/pprc/



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A.1.4

State sues informant in dumping probe

Cleanup of South Side dump sought

By Ray Gibson
TRIBUNE STAFF WRITER

For the second time this month, the informant in the federal government's probe of illegal dumping and aldermanic corruption was sued for operating a hazardous waste dump.

The latest suit against undercover mole John Christopher and others was filed Wednesday by the Illinois attorney general's office. It seeks an immediate cleanup of the 20-acre site at 76th Street and Albany Avenue and asks for fines that could total millions of dollars.

Earlier this month, the City of Chicago filed a nearly identical lawsuit in Cook County Circuit Court involving the same illegal dump.

Both lawsuits also named as defendants several corporations and individuals, including Miles Berger, a former chairman of the Chicago Plan Commission and prominent developer; James Pielet, a Highland Park businessman; and a Summit auto recycling center owned by steel magnate Cyrus Tang.

Berger is one of the owners in a land trust that owns the 20-acre site. Pielet formerly owned Midwest Metallics, the Summit recycling center, now owned by Tang.

The Summit junk yard allegedly shipped tons of hazardous waste to the 76th Street site for burial, and the lawsuit accuses Christopher of illegally dumping at the site beginning in 1989. He became a government informant in July 1992.

State and federal environmental officials contend that the buried material at the site contains lead and PCBs in concentration levels that are considered hazardous.

Attorneys for Pielet have contended that he never used the South Side dump.

A spokesman for Midwest Metallics said the dumping at the site predates Tang's ownership of the junk yard.

Berger said he hadn't seen the lawsuit yet. "It is very unfortunate. I own 2½ percent of this property," he said, declining further comment.

Christopher was named in the lawsuit chiefly because "he has the history of the site and he knows what went on with whom," said Matthew Dunn, chief of the environmental division of the attorney general's office.

The government probe, Operation Silver Shovel, so far has resulted in guilty pleas by Aldermen Ambrosio Medrano (25th) and Allan Streeter (17th), to extortion charges. A third alderman, Jesse Evans (21st), has pleaded innocent and is scheduled to go on trial in February. Two former city inspectors have pleaded guilty to taking bribes as well.

A defunct south suburban trucking outfit, Tone Trucking, also was accused in the lawsuit of hauling the hazardous waste to the site but was not named as a defendant. Dunn said the company is under investigation.

Tone was owned by Anthony Barone, a former Chicago police officer and reputed crime figure.